A CASE OF PYÆMIA.

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Reprinted from the Montreal Medical Journal, December, 1895.

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A CASE OF PYÆMIA.[1]

WITH SUGGESTIONS AS TO POSSIBLE CAUSE FOR PRESENCE OF A PRE-SYSTOLIC MURMUR WITHOUT STENOSIS OF THE MITRAL VALVE.

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The history of this case is of considerable interest owing to the following conditions which were present:

1. The apparent insignificance of the seat of infection, and the extensive distribution of the secondary symptoms.
2. The presence of a high degree of suppurative myocarditis.
3. The absence of abscess formation in the lung.
4. The presence of a presystolic thrill and murmur without stenosis of the mitral valve.

J. D., aged 21 years, employed as an assistant cook, was admitted to the medical department of the Royal Victoria Hospital on the 26th of July, 1895, complaining of general pains, extreme weakness, and vomiting.

Though he had not been in good health during June and the greater part of July, yet he continued to do his regular work. On the 23rd of July he became acutely ill, the onset of the attack being characterized by chill, headache, pains in the back, nausea, and vomiting.

The history of the patient showed him to be a subject of articular rheumatism as early as his seventh year, with subsequent and frequent attacks of sore throat, as well as recurrences of arthritic manifestations. The last attack of rheumatism occurred in June, 1894. The convalescence therefrom was not satisfactory, as cardiac complications were already established.

Shortly after admission with the complaints enumerated above, the following condition of the patient was noted (July 27, 1895):

He was delirious and irritable, skin dry and hot, face flushed with slight cyanosis about the ears and lips; lips and tongue dry and

[1] Read before the Montreal Medico-Chirurgical Society, October 18, 1895.

brown; the abdomen showed no signs of distension; the spleen was not palpable; there was no increase of liver dulness. The skin over the face, trunk, and extremities, presented an eruption, petechial, papular, and at some points pustular. On the ulnar surface of the left forearm, near the lower third, was a raised, slightly reddened patch, 3 cm. in diameter, tender on pressure, somewhat boggy to the feel, and presenting about its centre a small area about the size of a pin-head, at which the skin appeared in the state of recent healing—a recently closed opening without scab-thickening. This region on the forearm was said to be the seat of an injury received a few days before the onset of his illness. The right ankle showed on its anterior and inner aspect signs of a recent scald. It was partly healed and no signs of extending inflammation were seen. At the root of the nail of the right fore-finger there were signs of localized inflammation. The temperature at this examination was 103°, the pulse 120, respirations 30.

On examination of the circulatory system the signs present led to a diagnosis of cardiac hypertrophy due to valvular disease. The murmurs present indicated mitral incompetency with stenosis, as in addition to the apical systolic murmur which was transmitted to the axilla, a presystolic thrill and murmur were detected. In addition, distinct evidence of aortic valvular disease was manifest, inasmuch as both collapsing pulse and a diastolic murmur were observed.

The respiratory system showed, besides increase in the rate of respirations, diminished expansion over the upper half of the left side. There was no dulness on percussion. A few moist râles with diminished breath sounds were heard at the right base.

The urine was passed involuntarily. Its reaction was acid. Albumen was present.

The blood examination revealed the presence of a leucocytosis of the polynuclear variety, five to fourteen white cells being visible in each field. On the 27th and 28th of July cultures were made with agar and also with broth, each day giving the staphylococcus pyogenes aureus.

Progress of the case.—This was rapidly worse. The delirium of the 27th deepened into coma on the evening of the 28th July. The pulse rate increased to 160, while the strength of the beat diminished. The cardiac area was observed to increase toward the left by about one-half an inch. The respirations, 24 on admission, ran as high as 60 per minute on the 29th. The cyanosis became more marked. The temperature followed an irregular remittent curve, ranging from 100·6° to 105·8°. The signs in the lungs were those indicating œdema, with possibly an area of infiltration at the right base posteriorly.

On the 27th and 28th the movements of the left ankle, left great toe, left shoulder, and of the right little finger were painful, and redness and swelling were present over the ball of the left great toe, as well as over the dorsal surface of the little finger of the right hand. There was no diarrhœa nor vomiting during the progress of the case in the hospital.

On the 29th of July, the seventh day of the disease in its acute manifestations, the patient's condition was much worse, and he died at 3.30 p.m.

The question of diagnosis in this case needed not a little consideration to decide. Evidently we had to deal with an infection which was extremely active and virulent. Malignant endocarditis, typhoid fever, pneumonia, miliary tuberculosis, and pyæmia, at first, were all in the category of possibilities.

It was known that the patient was a subject of chronic endocarditis, a suitable condition for the ulcerative form to succeed, but what was the source, and where the entrance of the micro-organism? Malignant endocarditis could not be excluded. Indeed it appeared to be, in all probability, a part of the case.

The rapidity of the onset, the absence of abdominal signs, and the presence of leucocytosis, as well as the character of the eruption, were all against a diagnosis of typhoid fever.

For pneumonia, there were not physical signs sufficient to account for the condition of the patient.

An exclusion of miliary tuberculosis would, doubtless, have been impossible had not positive evidence of another disease been observed.

From the following features of this case, then, a diagnosis of acute pyæmia with, in all probability, multiple abscesses throughout the organs, was made: An injury with evidences of wound of skin surrounded by an area of tenderness and swelling; sudden onset of the illness; rapid advance of the case; albuminuria; hæmorrhagic eruption; joint redness and swelling; leucocytosis of an inflammatory character, and the presence of the staphylococcus pyogenes aureus in the blood.

The following is an abstract from the report of the autopsy performed four hours after death.

The body was that of a well-nourished and well-built young man, presenting the usual signs of death.

On the outer side of the fifth finger of the right hand there was a slightly reddened swelling, which on section presented a small drop of pus lying about the sheath of the extensor tendon. No abrasion appeared externally.

Upon the ulnar surface of the left forearm was seen a bluish-purple swelling 3 cm. in diameter, the centre of which showed externally a point of recent healing. Incision into this allowed the exit of greyish-green pus, situated amid much disin-

tegrated sloughy tissue, while the overlying skin, on the other hand, was much thickened and firmer than normal. The vessels in the neighbourhood were free.

Examination of the various joints and corresponding veins was negative. The abdominal cavity was dry, the visible coils of intestine distended and injected; in various parts of the serosa, were patches of a red and yellow colour corresponding to abscesses formed in and about the vessels of the serosa. The peritoneum otherwise seemed free from inflammation.

The spleen was large and soft and showed numerous miliary abscesses and some necrotic infarcts of small size.

The kidneys were large, of flabby consistence; the capsule, which stripped with slight difficulty, revealed a mottled red and yellow smooth surface upon which were some large anæmic infarcts and numerous small and large abscesses. On section, the cortex presented many miliary abscesses, chiefly about the vessels, though here and there septic sequestra were visible in the papillæ.

The pelvis and ureters were unaffected.

In the intestines there was further evidence of systemic infection, as above described, and also enlargement of the lymphoid structures. Here and there the abscesses had ulcerated through the mucosa, thus causing irregular ragged ulcers of the intestinal wall. The liver was enlarged, showed parenchymatous degeneration, and localized areas of necrosis. Many abscesses, small and large, were distributed throughout the organ, the largest about 3 to 4 cm. in diameter. In the lungs brown induration was the chief pathological condition. No abscesses whatever could here be found.

The heart was of considerable interest. Its pericardial surfaces were rather firmly adherent, so that in their separation much force was necessary, the adhesions being most firm over the anterior septum. Both layers were much thickened, the cavity dry, all the chambers of the heart very greatly hypertrophied and dilated. Subpericardial hæmorrhages and a few foci of suppuration were observed. The tricuspid orifice readily admitted four fingers, the mitral two, while three fingers were inserted with but slight difficulty. The mitral and aortic valves showed great chronic thickening, as did also the tendinous cords and papillary muscles. Upon the mitral, tricuspid, and aortic valves were greenish-grey friable vegetations of a malignant nature, those on the aortic valves being greatest in amount. On removal of these vegetations some loss of endocardial substance was evident. The pulmonary valves were free.

Further, in all the chambers, though especially in the ventricles, there were miliary subendocardial abscesses of the size of pin-heads dotted throughout the surfaces and each seemingly surrounded by a hæmorrhagic zone. Deep incision into the myocardium showed it to be beset with similar punctiform abscesses, also evidently embolic in nature.

The surface of the brain also showed abscesses of small size beneath the pia, causing loss of cerebral substance to a slight extent.

The cord was congested and œdematous, though so far as examined presented no abscesses.

The aural and nasal cavities appeared free from disease; the petrous bones normal.

Cultures taken from the blood, the skin wounds, and from all the other organs gave the same results, viz., in each case a copious growth of the staphylococcus pyogenes aureus.

Microscopic examination of the various tissues and organs merely verified the microscopic appearances, and hence is of comparatively little additional interest.

Our clinical conclusions, based both upon the clinical observations during the course of the malady and at the post-mortem examination, have led us to regard the case as one of pyæmia following upon a slight abrasion on the forearm. The date of injury, the amount of

disintegration of tissue out of all proportion to that elsewhere found; further, the inflammatory thickening of the overlying subcutaneous tissue, and the absence of any other visible seat of entry would in all probability warrant such an opinion. Without such an origin it would be almost impossible to do otherwise than regard the case in the light of the somewhat dubious cryptogenetic or so-called idiopathic pyæmias.

As possible objections to our view it may be urged that in the forearm we have merely an additional secondary focus, similar to all the others; yet not only is the condition apparently one of greater duration than the other affections, but further, the wound having had at one time definite connection with the outer air would hardly suggest a secondary focus. That no thrombo-phlebitis was found in the proximal vessels is indeed unusual, yet that the affection may occur without such a coincident event seems to us quite possible.

Nor is the case one of primary malignant endocarditis, for the valvular lesion is of the most acute type, one eminently recent and apparently contemporaneous with the other secondary events of the disease—the old valve lesions rendering this site a *locus minoris resistentiæ*.

Additional features of interest exist in the facts that three different sets of valves were affected, and that although the liver presented ample evidence of infection, the lungs, so commonly involved, showed here no signs of abscess formation. There is another point of no little interest to the clinician, in that the signs observed in the heart and the subsequently found pathological condition fail to correspond, and another example of the difficulty in exact diagnosis of cardiac conditions is afforded.

As described above, among the physical signs present on examination of the heart was a rough murmur, presystolic in rhythm, and well localized to the apex region—in other words, a combination of symptoms very suggestive of mitral stenosis. Yet at the autopsy the auriculo-ventricular orifice of the left side was of normal size, so that it would seem necessary to find some cause other than a mitral lesion to explain the presence of so typical a presystolic murmur. Within a recent date not a little discussion has arisen upon the origin of such conditions as those found in this case, and since Hunt[1] offered the classical suggestion that an aortic regurgitation may induce presystolic murmurs by bringing about an abnormal vibration of the mitral valves, further opinions have been elicited. Thus,

[1] Hunt (Austin), *Amer. Jour. Med. Science*, 1886. Vol. I, p. 27.

Theodore Fisher[1] has cited more than a dozen cases in which an adherent pericardium seemed a possible etiological factor, while Osler[2] and Graham Steele[3] have similar cases on record. A very recent thesis by Phear[4] has volunteered yet another explanation, that a thickening of the tendinous cords and dilatation of the ventricle may cause the two cusps of the mitral valve to be abnormally approximated and thus induce a functional stenosis.

In one of Professor Adami's[5] cases with dilated heart and enlarged mitral orifice there had been observed clinically a presystolic thrill and murmur. Although in our own case there had been present both adherent pericardium and aortic incompetence, it would seem that still another suggestion might be submitted. The heart being enlarged to nearly twice its normal size and the chambers presenting extreme dilatation, the quantity of blood contained by them is correspondingly increased. Under such circumstances there would be a very much greater amount of blood endeavouring to force its way through the mitral orifice, especially with the great hypertrophy seen in the auricle. Hence with a normal sized left auriculo-ventricular opening we have an excessive amount of blood seeking passage through it—in other words, the proportion of blood to the size of the passage makes a relative stenosis of the orifice. It may be further mentioned that in the majority of cases which present a similar set of conditions, very great dilatation has been present, and it would seem that according to the degree of dilatation, that is the amount of blood within the chambers, so there would be a presystolic murmur or not, this sign presenting only when the amount of blood is extreme.

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[1] Fisher, *B. M. J.*, April 23, 1894.
[2] Osler, *Trans. Ass. Amer. Phys.*, 1889. Vol. III., p. 138.
[3] Steell (Graham), *Practitioner*, April, 1894.
[4] Phear, *Lancet*, September 21, 1895.
[5] Adami, *Mont. Med. Journal*, April, 1895, p. 789.